LIVER CIRRHOSIS & ITS COMPLICATION

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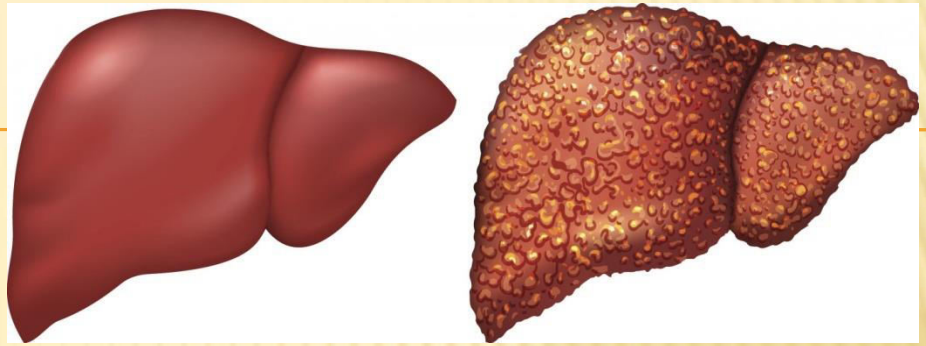
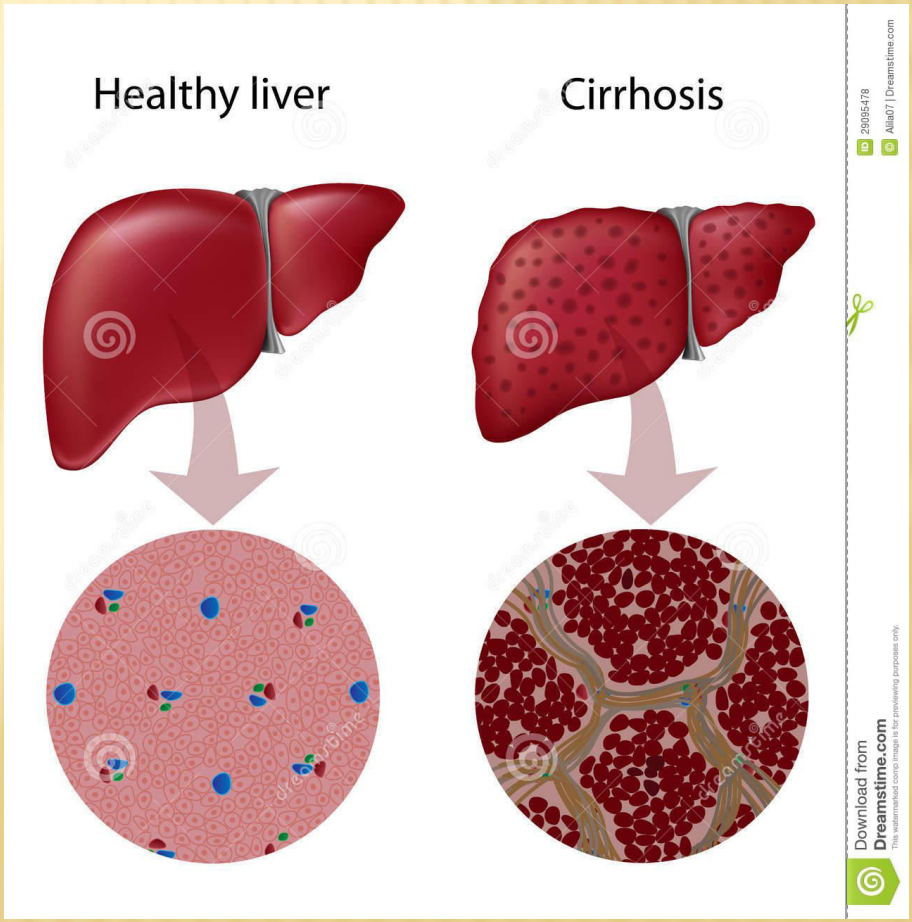
DEFINITION



- It is a **diffuse irreversible** process characterized by **necrosis** of liver cells, followed by **fibrosis**, **regeneration** and **nodule** formation.
- The liver architecture is diffusely abnormal and this interferes with liver blood flow and function. This finally leads to impaired liver function.



CAUSES 1

IT IS CAUSED BY CHRONIC NON SELF LIMITED INJURY OF THE LIVER

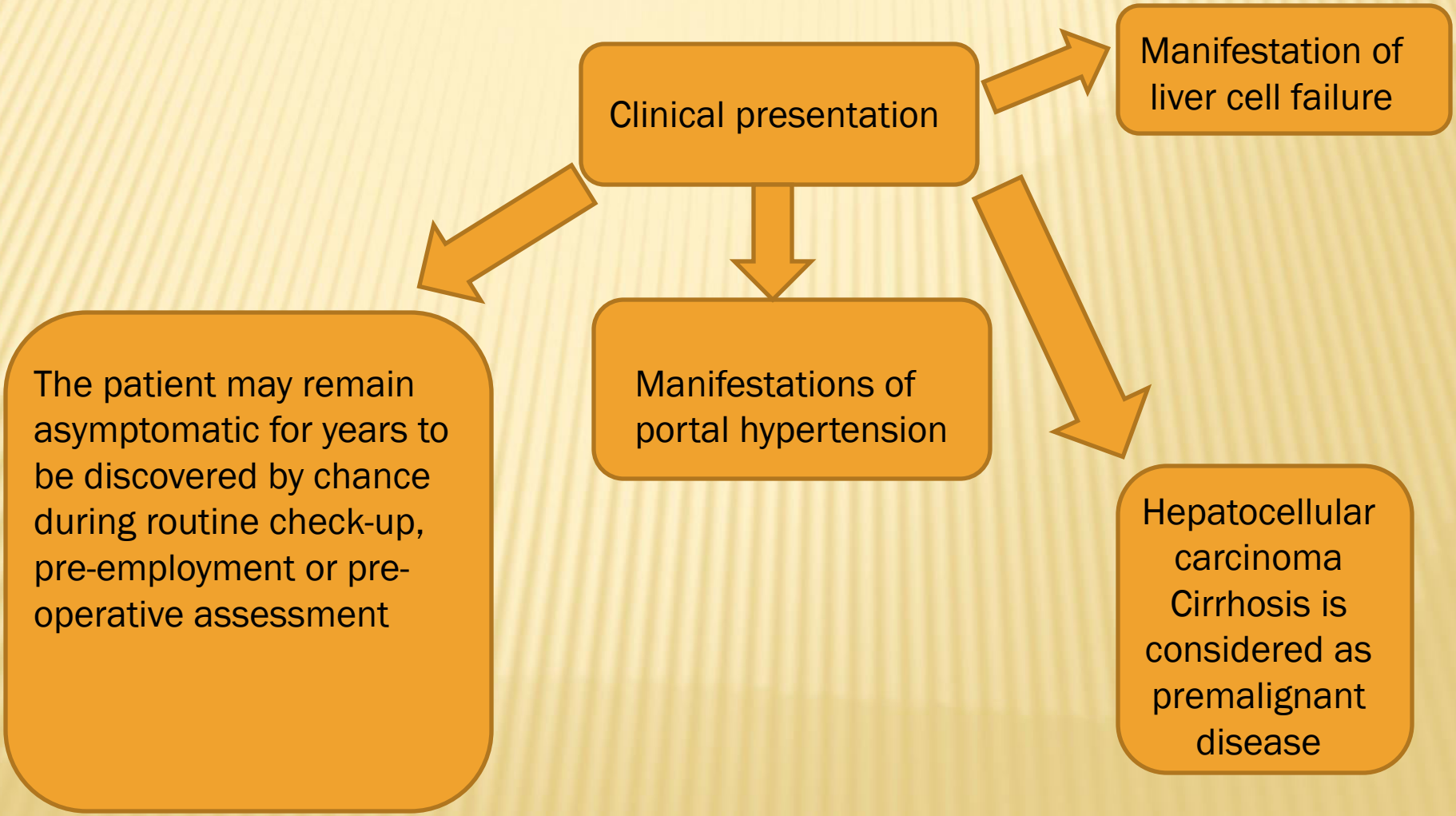
Viral hepatitis	Chronic hepatitis C Chronic hepatitis B±D	
Drug induced	Methotrexate Alpha methyl dopa Isoniazide	
Non-alcoholic steatohepatitis (NASH)	As a part from metabolic syndrome (associated with obesity, type 2 diabetes mellitus, dyslipidemia .....etc)	
Alcoholic steatohepatitis (ASH)	Prolonged (>10 years) Regular alcohol intake (>30 ml/day)	
Autoimmune hepatitis	The body's immune system attacks the hepatocytes, causing inflammation , damage and eventually cirrhosis.	

CAUSES 2

IT IS CAUSED BY CHRONIC NON SELF LIMITED INJURY OF THE LIVER

Hepatic congestion	•Prolonged right side heart failure. •Hepatic vein obstruction e.g. Budd chiari syndrome	
Metabolic disorders	• Haemochromatosis: Genetic defect leads to over absorption of iron from GIT and deposition in different tissues including liver. •Wilson's disease: Genetic defect in copper metabolism leads to failure of hepatic excretion of copper in bile → deposition in hepatic tissue. •Alpha-1 antitrypsin deficiency: failure of secretion of this enzyme and its accumulation in liver leads to damage of hepatocytes.	
Prolonged cholestasis	•Primary: e.g. Primary biliary cirrhosis. •Secondary: Primary sclerosing cholangitis or any prolonged biliary obstruction.	

CLINICAL SPECTRUM OF LIVER CIRRHOSIS



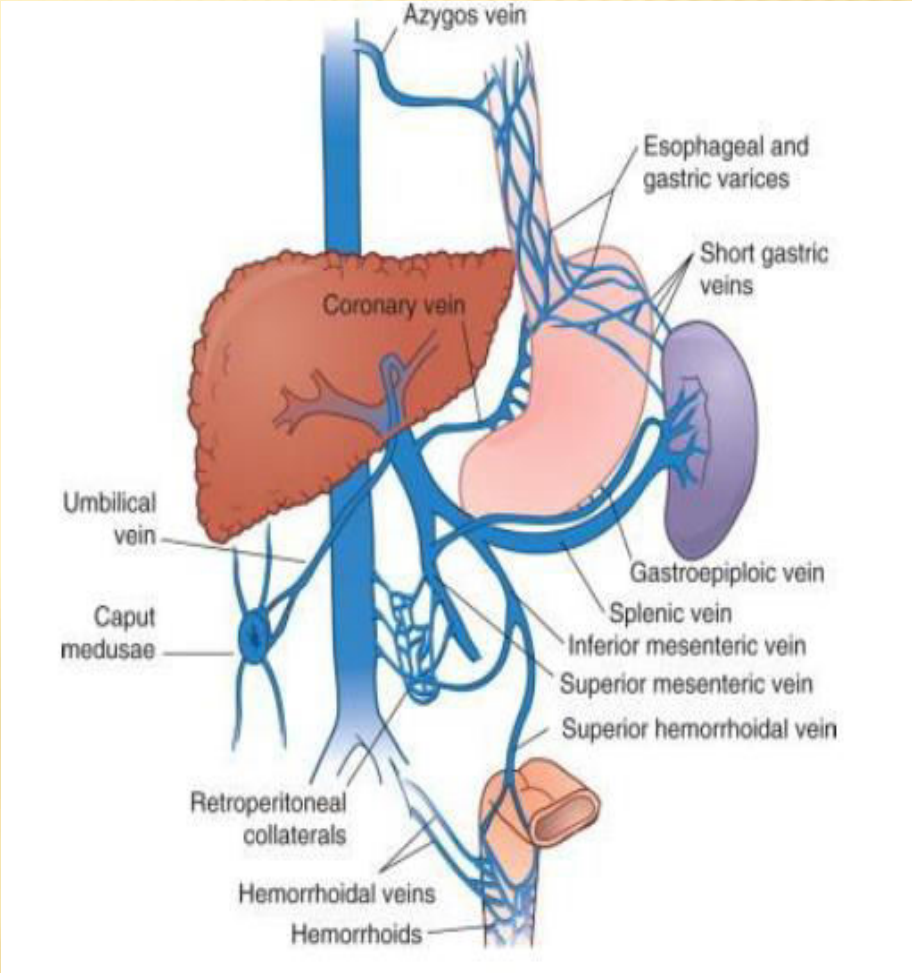
PORTAL HYPERTENSION1

- The portal pressure is normally 5-8 mmHg, and portal hypertension is considered when the pressure is more than 12 mmHg.
- Liver fibrosis may lead to obstruction/resistance to the inflow of blood in the portal venous radicles → leads to:

1- formation of portosystemic collaterals which bypass the liver and divert portal blood flow to systemic circulation.

2- Congestion & enlargement of the spleen.

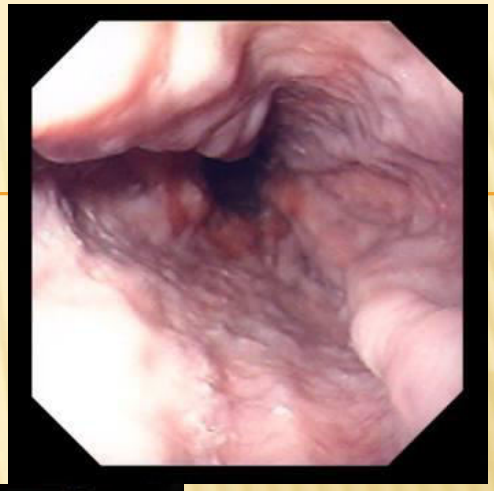
- The most common cause is: cirrhosis and schistosomiasis in endemic areas like Egypt.

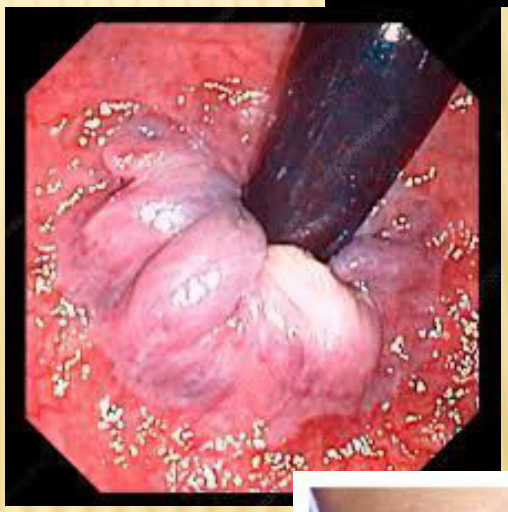


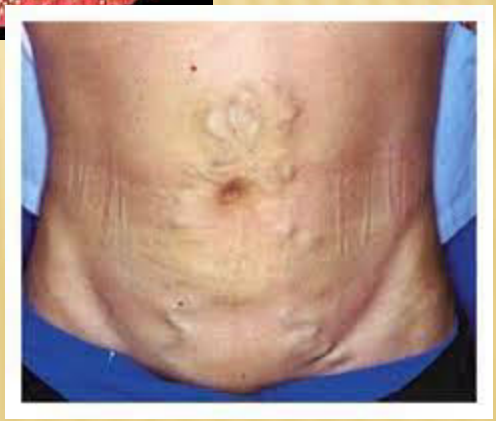
PORTAL HYPERTENSION2

- **The main sites of collaterals** are:

1. **At the gastro-esophageal junction (esophageal varices):** dilated veins at the lower end of the esophagus which may be associated with gastric varices in the fundus of the stomach. They develop into anastomosis between left and short gastric veins (portal) and azygos vein (systemic). Esophageal varices are superficial in position and tend to rupture.
2. **Rectal varices:** due to anastomosis between superior haemorrhoidal (portal) and middle and inferior haemorrhoidal (systemic).
3. **At the anterior abdominal wall: (caput medousa)** due to recanalisation of the umbilical vein (portal) with the superior and inferior epigastric veins (systemic).
4. **Others**: retroperitoneal collaterals & lineo-renal collateral.

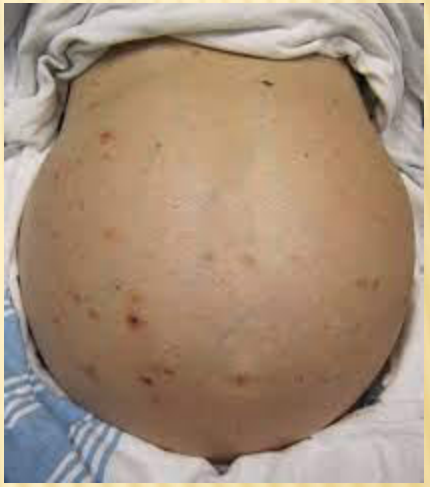


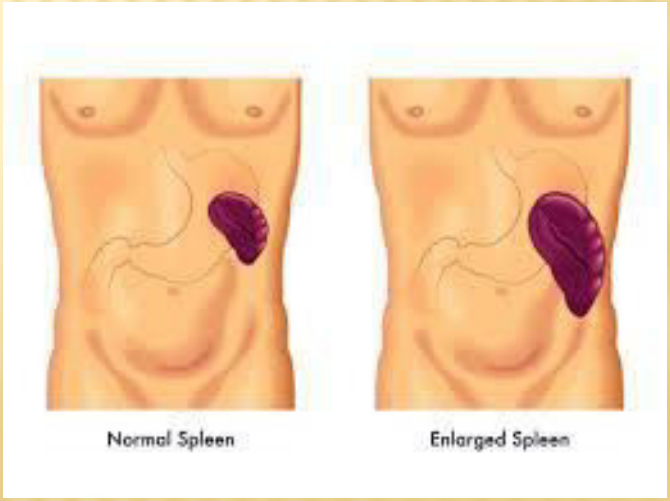




PORTAL HYPERTENSION 3 (CLINICAL PICTURE)

- Splenomegaly: due to splenic vein congestion + reticulo-endothelial hyperplasia→ hypersplenism → thrombocytopenia.
- Haematemesis and melena from rupture of gastro-esophageal varices or congestive gastropathy.
- Bleeding per rectum: haemorrhoidal bleeding.
- Encephalopathy due to passage of toxins through portosystemic shunts.
- Dilated para umbilical veins
- Nausea and anorexia (due to congestion of gastrointestinal tract
- Ascites (multifactorial):





CHRONIC LIVER CELLFAILURE 1

Degeneration of the liver cells may result in:

1. **Hypoalbuminaemia:** failure of the diseased liver to synthesize albumin → Low plasma albumin → lower limb oedema & ascites.

N.B. Causes of ascites in liver cirrhosis:

- Hypoalbuminaemia.
- Portal hypertension: increased hydrostatic pressure→ fluid leaking into peritoneum.
- Renal salt & water retention (secondary hyperaldosteronism).

CHRONIC LIVER CELLFAILURE 2

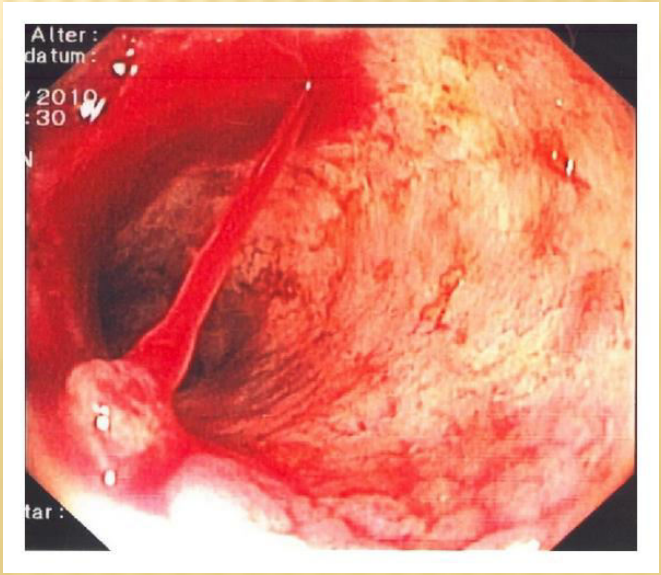
2. **Jaundice:** due to inability of the liver cells to uptake & secrete bilirubin.
3. **Coagulopathy:** Increased bleeding tendency due to defective synthesis of coagulation factors (II, VII,IX and X) by the failing liver.
4. **Hepatic encephalopathy:** Neuropsychiatric syndrome that results from failure of the liver cells to detoxify nitrogenous substances produced in the colon (ammonia). These substances reach the brain (through portosytemic shunts) resulting in:

    a. Changes in personality

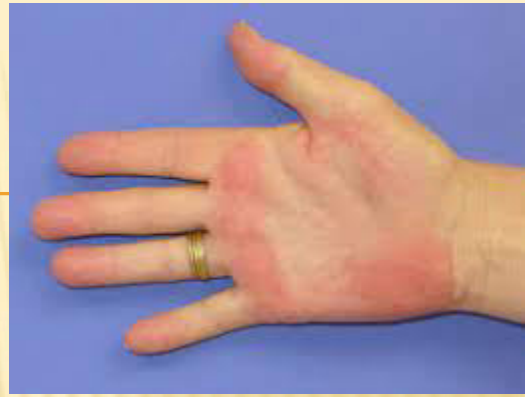
    b. Intellectual deterioration

    c. Tremors in hands

    d. Later, terminal hepatic coma



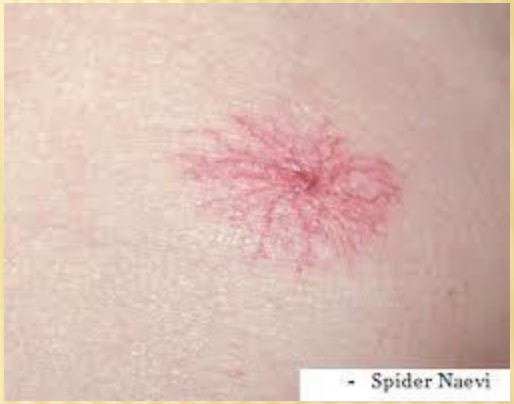
CHRONIC LIVER CELLFAILURE 3

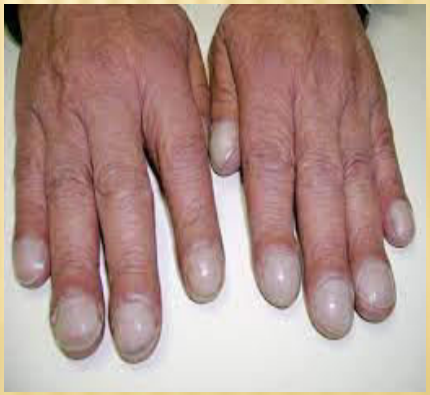


5. **Endocrinal manefistations:**

Of unknown cause; but may be due to failure of liver to inactivate circulating estrogens with the following observations:

- Palmar erythema: reddish discoloration of the skin opposite the thenar, hypothenar and head of metacarpal bones.
- Spidar angiomata (naevi): in skin of face, arms and hands (dilated arteriole with radiating capillaries).
- Gynacomastia, female distribution of hair, impotence and testicular atrophy in males.
- Amenorrhoea, and sterility in females.





6. **Clubbing:** pale clubbing.
7. **Foetor hepaticus**: bad mouth breath due to appearance of nitrogenous substances absorbed from colon and either not detoxified by the failed liver or reach circulation via opened collaterals.

CAUSES OF DEATH:

1. Severe attack of Haematemsis.
2. Hepatic encephalopathy.
3. Hepatorenal syndrome: functional renal failure occurs in terminal patients with liver cell failure, the kidney histology is normal.
4. Hepatocellular carcinoma.
5. Sepsis.

IMPORTANT NOTE

- Hepatic schistosomiasis doesn't cause liver cirrhosis.
- Schistosomal hepatic affection leads to hepatic fibrosis and portal hypertension.

INVESTIGATIONS

- **CBC:**
  - Anaemia: due to bleeding.
  - Thrombocytopenia: hypersplenism.

- **Liver function tests:**
  - Liver enzymes: mild to moderate elevation of ALT & AST.
  - Bilirubin: increased (biphasic).
  - Albumin: decreased.
  - Prothrombin time: prolonged.

- **Investigations of the cause: e.g. viral markers, liver biopsy.**

- **Alpha-feto protein:**

tumor marker should be ordered every 6 months for early detection of HCC.

- **Abdominal ultrasound:**
  - Liver: shrunken & cirrhotic.
  - Spleen: enlarged & congested.
  - Ascites.
  - Cheap & easy method for detection of hepatic focal lesions.

TREATMENT

- Hepatic fibrosis التليف(stages precedes cirrhosis) is a reversible process if the etiologic agent is corrected.
- Hepatic cirrhosis التشمع is an irreversible condition, has no treatment.
- Treatment lines are dedicated for controlling complications only.
- Liver transplantation is the only current hope for improving survival and better quality of life.

- Examples:
  - Ascites: decrease salt intake, diuretics and tapping in severe cases.
  - Endoscopic band ligation for bleeding varices.